Exploring neural markers of incentive salience and real-world drinking among individuals with alcohol use disorder.

***Dahyeon Kang, Ph.D.[a, 1], *Silvia Murgia, Ph.D.[a], Eddie P. Caumiant[a], Zoe Lee[a, 2], Walter J. Venerable, III, Ph.D.[a, 3], Alexa Boland[a, 4], Catharine E. Fairbairn, Ph.D.[a], and Kara D. Federmeier, Ph.D.[a, b]**

[a] Department of Psychology, University of Illinois at Urbana-Champaign, Champaign, IL 61820, USA

*Dahyeon Kang, email: danikang@uw.edu; *Silvia Murgia, email: smurgia2@illinois.edu; Eddie P. Caumiant, email: eddiepc2@illinois.edu; Zoe Lee, email: zl6@fordham.edu; Walter J. Venerable, III, email: walter.venerable@outlook.com; Alexa Boland, email: aboland3@kent.edu; Catharine E. Fairbairn, email: cfairbai@illinois.edu; Kara D. Federmeier, email: kfederme@illinois.edu

[b] Beckman Institute for Advanced Science and Technology, University of Illinois at Urbana-Champaign, USA

Author Note

* Shared first authorship; the first and second authors contributed equally to this paper.

Correspondence concerning this article should be addressed either to Silvia Murgia, Ph.D., Department of Psychology, University of Illinois—Urbana-Champaign, 603 East Daniel St., Champaign, Illinois, USA, 61820, electronic mail: smurgia2@illinois.edu, or to Catharine Fairbairn, Ph.D., Department of Psychology, University of Illinois—Urbana-Champaign, 603 East Daniel St., Champaign, Illinois, USA, 61820, electronic mail: cfairbai@illinois.edu.

---

[1] Present address: Department of Psychiatry and Behavioral Sciences, University of Washington School of Medicine, Seattle, WA, 91801, USA
[2] Present address: Department of Psychology Fordham University, New York City, NY 10458, USA
[3] Present address: Addiction Recovery Treatment Services, McClellan VA Outpatient Clinic; McClellan Park, CA 95652, USA
[4] Present address: Department of Psychological Sciences, Kent State University, Kent, OH 44240, USA

Abstract

Alcohol cue salience is theorized to play a mechanistic role in alcohol use disorder (AUD), yet links between neural cue reactivity and naturalistic drinking remain undercharacterized. This study combined laboratory event-related potential (ERP) measures with two weeks of ambulatory assessment to evaluate whether alcohol-cue P3b relates to real-world drinking and individual differences in AUD severity. Heavy drinking participants (52% Female; Ages 21-32) were recruited from the local community. Participants completed two weeks of ecological momentary assessment with continuous transdermal alcohol monitoring and attended three laboratory visits scheduled at one-week intervals. At the final study visit, participants completed an EEG visual oddball task involving the presentation of both infrequent alcohol and non-alcohol beverage target images and frequent household-object standards. Alcohol images elicited significantly larger P3b amplitudes than non-alcohol images across the sample ($N = 47$), $b = 2.13$, $p = .002$. Critically, this alcohol-specific P3b enhancement was concentrated among individuals with pronounced AUD (moderate–severe, N=20). Objective transdermally-measured ambulatory drinking further moderated neural cue reactivity in the pronounced AUD group: more binge-level days, b=0.51, p=.020, and higher peak estimated consumption, b=34.09, p=.044, were associated with stronger alcohol-specific P3b responses. In contrast, neither retrospective baseline nor in-vivo ambulatory self-reports of drinking demonstrated consistent associations. Together, findings indicate that alcohol cue–elicited P3b is (a) sensitive to clinically meaningful severity distinctions and (b) larger among individuals with heavier real-world drinking as captured with objective sensors, supporting its utility as a neurocognitive marker with ecological validity for understanding individual differences in AUD.

Keywords: Alcohol Use Disorder; ERP; P3b; alcohol cue salience; ambulatory assessment; transdermal alcohol monitoring.

1. Introduction

Addiction researchers have long stressed the importance of better understanding heterogeneity in alcohol use disorder (AUD) phenotypes, as such variability may help explain why individuals respond differently to specific treatment modalities (Witkiewitz et al., 2019). Consistent with this view, research has repeatedly documented meaningful differences among individuals with AUD in their alcohol-use patterns, psychosocial characteristics, and neurobiological signatures (Litten et al., 2015). To organize this variability, the Alcohol Addiction Research Domain Criteria (AARDoC) extends the National Institute of Mental Health RDoC framework to addictions, delineating three core research domains—i.e., incentive salience, negative emotionality, and executive function—to advance understanding of AUD etiology, course, and treatment (Kwako et al., 2016; Koob & Volkow, 2016). Building on this framework, the present work centers on incentive salience for alcohol cues and its relevance for real-world drinking among people with AUD.

Incentive salience refers to psychological processes that assign motivational significance to potentially rewarding stimuli (Paulus et al., 2009; Robinson et al., 2014; Robinson & Berridge, 1993). Within addiction theory, incentive salience toward alcohol-related stimuli, or *alcohol cue salience*, has been advanced as a core mechanism of addiction (Carter & Tiffany, 1999; MacKillop & Lisman, 2008; Niaura et al., 1988; Robinson & Berridge, 1993). Alcohol cue salience varies markedly with individual and contextual characteristics; depending on the person and the setting, a can of beer or a glass of wine might exert little sway over awareness or, alternatively, may seem to leap out at the drinker and dominate attention (Martins et al., 2019; Valyear et al., 2017; Villaruel & Chaudhri, 2016). Heightened alcohol cue salience has been identified as a potent immediate precipitant of drinking (Kambouropoulos & Staiger, 2009) and,

over time, is implicated in the development of AUD (Cox et al., 2002; Robinson & Berridge, 1993; Stormark et al., 1997). Consequently, characterizing alcohol cue salience has become a priority in addiction science.

Efforts to capture alcohol cue salience have faced notable methodological challenges because the attentional operations that prioritize environmental cues often unfold outside conscious awareness (Krank & Wall, 2006), limiting the interpretability of self-report alone. Methods that index neural activity on a millisecond timescale, particularly event-related brain potentials (ERPs), offer an opportunity to observe these rapid, automatic processes directly. Of special relevance is the P3b, a positive ERP component that peaks about 300 ms (or later) following stimulus onset and is largest over posterior scalp sites. It is most commonly elicited in oddball paradigms, in which infrequent target stimuli are presented among frequent standard stimuli. P3b amplitude scales with subjective probability, such that rarer events (e.g., oddballs) elicit larger responses, and, critically, is further modulated by the amount of attention allocated to the stimulus (Donchin, 1981; Polich & Kok, 1995; Polich, 2007). In alcohol-cue paradigms that intermix pictures of alcohol and non-alcohol beverage targets among neutral standards, the amplitude difference between P3b responses elicited by alcohol versus non-alcohol images has been used as an index of alcohol cue salience (Porjesz et al., 2005; Bartholow et al., 2007, 2010, Kohen et al., 2024). Across multiple studies, this alcohol cue salience marker – i.e., larger amplitude P3b responses to alcohol compared to non-alcohol targets – has been found to covary with individual and contextual factors linked to consumption and problem drinking (e.g., Bartholow et al., 2007; Herrmann et al., 2001; Namkoong et al., 2004; Bartholow et al., 2010; Cofresí et al., 2019; Fairbairn et al., 2021).

Lab-to-field findings indicate that laboratory measures of alcohol-cue processing, particularly alcohol-cue-elicited P3b responses, carry predictive relevance for drinking behavior in everyday life. For example, Kohen et al. (2024) reported that larger alcohol-cue–elicited P3b amplitudes forecast steeper real-world increases in alcohol consumption during drinking episodes and higher momentary craving across a 21-day EMA period, strengthening the case that laboratory P3b cue-reactivity tracks naturalistic drinking dynamics. More broadly, findings indicate that stronger neural prioritization of alcohol-related information is mirrored by heavier and more intense drinking patterns outside the laboratory, lending support to the mechanistic significance of incentive salience within the AARDoC framework (Kohen et al., 2024; Cofresí et al., 2022; Petit et al., 2015).

Complementary advances in wearable transdermal alcohol sensing and EMA methodology have improved the precision with which naturalistic drinking can be quantified, allowing more direct tests of how neural cue-reactivity relates to real-world alcohol exposure and subjective responses (Fairbairn & Bosch, 2021; Fairbairn, et al., 2020; Didier et al., 2024; Ariss et al., 2023). Consideration of individual-level variation is central to connecting alcohol-cue P3b with clinically meaningful outcomes. Although individuals with AUD frequently exhibit reduced P3b amplitudes to simple, nonaffective stimuli, consistent with deficits related to chronic heavy use and externalizing vulnerability (Euser et al., 2012; Patrick et al., 2006), several studies indicate enhanced P3b responses to alcohol-related images among individuals with AUD compared with social drinkers (Genkina & Shostakovich, 1983; Herrmann et al., 2000). In research employing a visual oddball paradigm, Namkoong et al. (2004) reported that social drinkers produced comparable P3b responses to alcohol and non-alcohol beverage images, whereas individuals with AUD produced larger P3b amplitudes to alcohol cues relative to non-

alcohol cues, a pattern replicated in later work (Littel et al., 2013; Petit et al., 2015). Related evidence suggests that nonclinical, at-risk drinkers may also show heightened P3b alcohol cue salience, potentially marking elevated vulnerability: heavier or riskier drinking and family history of alcoholism have each been associated with larger P3b's to alcohol versus non-alcohol cues (Herrmann et al., 2001; Ehlers et al., 2003; Bartholow et al., 2007, 2010; Petit et al., 2013, 2014).

However, most prior studies have not consistently yielded continuous associations between alcohol-cue P3b magnitude and retrospective self-reported consumption (Cofresí et al., 2019; Littel et al., 2012).. Modest sample sizes have further limited precision and generalizability (Cofresí et al., 2019). These gaps highlight the need to evaluate alcohol-cue P3b as an individual-difference construct across participants evincing a broad range of AUD severity levels while also considering clinically meaningful severity distinctions and linking laboratory indices to drinking as it occurs in daily life using modern ambulatory approaches (Fairbairn & Bosch, 2021; Shiffman et al., 2008).

In response to these needs, this study used a combined laboratory–ambulatory design among a sample of individuals with varying alcohol disorder symptom profiles, presenting with AUD severity levels spanning from "none/mild" to "severe." Participants completed a two-week ambulatory monitoring period (EMA paired with continuous transdermal alcohol monitoring) anchored by three laboratory visits scheduled one week apart, with the electroencephalography (EEG) oddball task administered at the final visit. Alcohol-cue salience was indexed by P3b amplitudes elicited by alcohol and non-alcohol beverage targets during a standard image-viewing oddball task, with the alcohol – non-alcohol difference treated as a putative neural marker of incentive salience (Porjesz et al., 2005; Bartholow et al., 2007, 2010). Complementing

retrospective assessments of drinking frequency, quantity, and alcohol-related problems, daily-life alcohol use and subjective responses were captured over two weeks using ecological momentary assessment (EMA) coupled with transdermal alcohol monitoring, providing objective, time-stamped estimates of consumption.

Within this framework, it is possible to test whether alcohol images elicit larger P3b amplitudes than non-alcohol images among clinically affected drinkers (Herrmann et al., 2000; Namkoong et al., 2004; Littel et al., 2013; Petit et al., 2015), to quantify dimensional associations between alcohol-cue P3b magnitude and severity indices (Armor et al., 1978; Fairbairn et al., 2018; Hasin et al., 2013; Kiluk et al., 2019; Mellentin et al., 2021), and to evaluate the ecological validity of the laboratory index through links to EMA- and sensor-derived markers of naturalistic drinking (Fairbairn et al., 2020; Fairbairn & Bosch, 2021). We hypothesized that, in this clinically affected sample, alcohol-related cues would elicit larger P3b responses than non-alcohol-related cues, that stronger P3b responses would be associated with heavier/more intense patterns of naturalistic drinking, that these associations would persist after accounting for conventional severity assessments, and that the strength of P3b–drinking links would be greater among individuals with higher overall severity and would align with momentary alcohol experiences.

2. Methods

2.1 Participants

Heavy drinking adults (*N*=60) of legal drinking age (≥21) were recruited from the local community. Exclusion criteria were: (a) self-reported formal diagnosis of a mental disorder (e.g., schizophrenia, bipolar disorder, major depressive disorder); (b) currently seeking treatment for AUD; (c) use of illicit drugs other than cannabis; and (d) history of traumatic brain injury or

brain surgery. The final sample comprised 31 males and 29 females with a mean age of 22.62 (*SD* = 2.88, range = 21-32). Regarding racial identity, participants comprised 39 White/Caucasian (65.0%), 5 Black/African American (8.3%), 13 Asian (21.7%), 1 American Indian/Alaska Native (1.7%), 1 Native Hawaiian/Other Pacific Islander (1.7%), and 1 multiracial (1.7%) individual. Twelve participants (20.0%) identified as Hispanic. All participants were required to meet initial DSM-5 criteria for alcohol use disorder according to a brief phone-based symptom checklist (American Psychiatric Association, 2013; Hasin et al., 2013). The descriptive characteristics of the sample are reported on Table 1. Recruitment targeted any community member who met eligibility criteria, and the final recruited sample comprised primarily students of variable levels (*N* = 6 part time; *N* = 52 full time). Handedness was not employed as a study exclusion; however, all participants in the final sample were right-handed. Study aims and hypotheses are registered at https://osf.io/m3puv.

2.2 Procedure

The study spanned two weeks and included three laboratory sessions scheduled one week apart (Figure 1; see also Ariss et al., 2025). All procedures were approved by the University of Illinois at Urbana–Champaign Institutional Review Board (Protocol No. IRB24-1785).

2.2.1 Session 1

The first laboratory session served as a study orientation. Participants were instructed to abstain from alcohol for at least 12 hours prior to this visit, and BrAC was verified to be 0.00% upon arrival. If BrAC was greater than 0.00% or participants verbally reported alcohol use within the past 12 hours, the study visit was rescheduled. Participants then provided informed consent, were fitted with a Secure Continuous Remote Alcohol Monitoring (SCRAM©) ankle bracelet, and the study smartphone application was installed on participants' smartphones. Through this

app, participants received eight smartphone surveys per day assessing alcohol use. Prompts fired at random within one of four 10-hour windows, 2:00 pm–12:00 am, 3:00 pm–1:00 am, 4:00 pm–2:00 am, or 5:00 pm–3:00 am, selected to match each participant's reported sleep schedule. During this session, participants also completed brief retrospective past-30-day quantity, frequency, and binge self-report drinking measures (see Measures for full description). Finally, formal AUD diagnostic status was assessed via the structured clinical interview of the DSM-5 administered by a research assistant (First et al., 2015; American Psychiatric Association, 2013).

2.2.2 Session 2

Approximately one week after the first visit, participants returned to the lab or met with researchers via a private Zoom session (~15 minutes) to troubleshoot any issues with the smartphone app or SCRAM device and address questions about the study.

2.2.3 Session 3

At the conclusion of the two-week ambulatory monitoring period, participants returned for a final laboratory session approximately two weeks after study initiation (Visit 1 to Visit 3: mean = 14.2 days; range = 13–19 days). During this session, participants completed an alcohol image-viewing oddball task while EEG was recorded. The task comprised three blocks of 76 images each (per block: 60 neutral context pictures; 8 alcohol-beverage pictures; 8 non-alcohol beverage pictures). All laboratory sessions were scheduled across the day, with Visit 1 start times ranging from 9:00 AM to 7:30 PM, Visit 2 from 9:00 AM to 7:30 PM, and Visit 3 from 10:00 AM to 8:30 PM. At session end, participants returned the SCRAM device and received $150, with an additional $50 contingent on ≥70% EMA compliance.

2.2.4 EEG Recording and Preprocessing

EEG was recorded from 4 Ag/AgCl scalp midline electrodes placed approximately at Fpz, Cz, Pz, and Oz (10–20 system, Jasper, 1958), referenced online to the left mastoid and re-referenced offline to the average of both mastoids; an additional peri-ocular electrode (between the left infraorbital ridge and the left outer canthus) monitored eye movements/blinks. Impedances were kept <5 kΩ. Signals were amplified (Sensorium amplifier) through a bandpass filter of 0.02-100 Hz and sampled at 250 Hz. Preprocessing was conducted using the *Event-Related Potential Software System* (Jon Hansen, UCSD). Data were baseline corrected using the −100 to 0 ms prestimulus interval and low-pass filtered at 30-Hz (3rd-order Butterworth). Artifact rejection (for blocking, drift, saccades, and blinks) was done via thresholds set for individual subjects following condition-blind visual inspection. On average 3% of trials were rejected from critical bins (range 0-46%). All critical bins contained a minimum of 12 trials (at least 50% of oddball trials). No electrode interpolation was performed.

2.2.5 Visual Oddball Task

A visual oddball task was used to present relatively infrequent alcohol and non-alcohol beverage target pictures among standards made up of neutral context pictures. The neutral context pictures[5] (60 total) were drawn from the International Affective Picture System (Lang et al., 2008) and included household objects such as a towel, a hairdryer, a cloth hanger, etc. According to Lang et al. (2008), these neutral context pictures had a mean valence rating of 5.06 (*SD* = 0.42) and a mean arousal rating of 3.03 (*SD* = 0.62) (1-9 scales). The alcohol beverage pictures (8 total) included depictions of beer bottles, a shot glass, a tequila bottle, a gin bottle, a rum bottle, a glass of wine, a pitcher of beer, and several types of alcohol-containing cocktails.

[5] IAPS numbers for the images: 7002, 7004, 7006, 7010, 7020, 7025, 7030, 7031, 7034, 7038, 7044, 7050, 7053, 7054, 7055, 7056, 7058, 7060, 7080, 7090, 7110, 7150, 7170, 7175, 7179, 7190, 7192, 7205, 7211, 7217, 7224, 7233, 7234, 7235, 7236, 5030, 5390, 5395, 5471, 5500, 5510, 5520, 5530, 5533, 5534, 5535, 5731, 7095, 7096, 7100, 7140, 7160, 7161, 7182, 7183, 7184, 7185, 7186, 7187, 7700.

The non-alcohol beverage pictures (8 total) included several types of juice in glasses (e.g., orange, apple, lemonade), a glass of milk, a cup of coffee, a bottle of water, and a sports drink. None of the beverage pictures contained people in order to prevent emotional responses that might confound participants' reactivity to beverage cues (Stritzke et al., 2004). All images were presented in the center of a 21" CRT computer monitor at a viewing distance of 100 cm. Images subtended 4.3 degrees of vertical visual angle and 6.5 degrees of horizontal visual angle.

The Alcohol Image Viewing Task consisted of 3 blocks, between which participants were given a short break (1-3 minutes). Each block contained the same set of 76 images presented in a different randomized order, with block order counterbalanced. Each trial began with a fixation display of three plus signs (+++) in the center of the screen for 1000 ms, followed by stimulus presentation for 1000 ms. Participants were instructed to respond to beverage targets by pressing a response button in one hand for an alcohol beverage picture or a button in the other hand for a non-alcohol beverage picture, while withholding responses to all other images. Response hand mapping was counterbalanced across participants. A 500 ms inter-trial interval followed stimulus offset. Participants were instructed to maintain central fixation throughout the experiment and to minimize eye blinks and facial movements.

2.3 Measures

2.3.1 P3b Alcohol Cue Salience

Oddball P3b effects are maximal over parietal scalp; thus, analyses focused on electrode Pz. Following Bartholow et al. (2007), mean amplitude was measured 300–800 ms post-stimulus-onset to accommodate individual variability in peak latency. For target trials (beverage images), only those with a correct response were included. Participant-level mean P3b amplitudes were measured for alcohol and non-alcohol targets and subjected to statistical

analyses looking at associations with clinical and ambulatory measures. Alcohol cue salience was operationalized as the difference in P3b amplitude between alcohol and non-alcohol target images, with larger positive values indicating stronger neural prioritization of alcohol cues.

2.3.2 AUD Severity Level

The Structured Clinical Interview for DSM-5 (SCID-5, First et al., 2015) defines AUD severity categories based on the number of endorsed criteria, with 2–3 criteria indicating mild, 4–5 indicating moderate, and 6–11 indicating severe AUD (Hasin et al., 2013). Importantly, distinctions between mild and the more clinically significant moderate and severe categories are well established (Kiluk et al., 2019; Mellentin et al., 2021). Consistent with prior ERP research, where binary groupings are often employed, a binary index of AUD severity was also used: 0–3 criteria reflecting “minimal” and 4–11 criteria reflecting “pronounced” alcohol problems.

2.3.3 Retrospective Self-Report of Typical Alcohol Consumption

During Session 1, participants completed a questionnaire derived from NIAAA’s recommended alcohol-use question sets (NIAAA, 2018) consisting of individual items assessing the frequency and quantity of alcohol consumption (Armor et al., 1978; Fairbairn et al., 2018). Items included: *1) Retrospective Self-Report of Drinking Frequency:* Participants were asked to report on how many days out of the past 30 they had at least one drink of alcohol; *2) Retrospective Self-Report of Drinking Quantity:* Participants were asked to report how many drinks they consumed on average per drinking day; *3) Retrospective Self-Report of Binge Drinking Frequency:* Participants indicated how often they engaged in binge drinking in the past 30 days (4+ standard drinks in a sitting for women, 5+ standard drinks for men).

2.3.4 Objective Transdermal Ambulatory Alcohol Consumption

The SCRAM ankle bracelet was used to measure participants' Transdermal Alcohol Concentration (TAC). In order to compute alcohol consumption levels for participants, readings derived from transdermal sensors were converted into estimated Blood Alcohol Content (eBAC) values using a machine learning model (Fairbairn et al., 2020). The machine learning algorithm employed to convert TAC into eBAC in the current analyses has been shown to produce values within 10% of true BAC in prior laboratory-based validation research (Fairbairn et al., 2020; see also (Fairbairn & Bosch, 2021). The following variables were created using the eBAC data: *1) eBAC Drinking Days*: The total number of days on which at least one eBAC value was higher than 0.01%; *2) eBAC Drinking Frequency:* The total number of eBAC readings higher than 0.01%, divided by the total number of eBAC readings during the ambulatory period; *3) eBAC Drinking Quantity*: For each drinking day, the mean of all eBAC readings ≥ 0.01%, then averaged across all drinking days (i.e., the day-weighted mean of positive eBAC); *4) eBAC Binge Drinking Frequency*: The total number of drinking days on which at least one eBAC value was higher than 0.08%; *5) eBAC Peak Average*: The highest eBAC recorded each day, averaged across the 14 days for each participant.

2.3.5 Self-Report Ambulatory Alcohol Consumption

Participants also provided self-reports of alcohol consumption during the ambulatory period (Shiffman et al., 2008; Stone & Shiffman, 1994). The following variables were created using the self-report data: *1) Ambulatory Self-Reported Drinking Days*: The total number of days on which the participant reported either currently drinking alcohol or having had at least one alcohol-containing drink since the last survey; *2) Ambulatory Self-Reported Drinking Frequency*: The total number of surveys the participant indicated drinking, divided by the total number of surveys submitted during the ambulatory period; *3) Ambulatory Self-Reported Drinking*

Quantity: The total self-reported number of drinks consumed over the course of the ambulatory period, divided by ambulatory self-reported drinking days; *4) Ambulatory Self-Reported Binge Drinking Frequency*: The total number of drinking days on which the total number of drinks consumed was 4+ (for women) or 5+ (for men), (National Institute on Alcohol Abuse and Alcoholism [NIAAA], 2004); *5) Ambulatory Self-Reported Peak day:* maximum number of drinks per participant on a single drinking day.

2.4 Data Analysis Plan

The data and code required to reproduce the analysis have been made publicly available at the Open Science Framework and can be accessed at https://osf.io/m3puv/files. All analyses were conducted in R (version 4.5.0) using mixed-effects models with the lme4 and lmerTest packages (Bates et al., 2015; Kuznetsova et al., 2017). Descriptive statistics were first computed to characterize the sample in terms of age, gender, and overall ERP amplitudes across conditions. The primary analyses focused on examining differences in P3b ERP amplitudes elicited by alcohol versus non-alcohol image cues. ERP outcomes were mean P3b amplitudes at Pz (300–800 ms) computed separately for alcohol and non-alcohol target trials per participant (correct-response trials only), yielding two condition-specific values per participant. Although the integration of random slopes was not possible given the dyadic nature of clustering in this sample, clustering was accounted for in the random intercept components (Kenny et al., 2016).

Stimulus type (alcohol vs. non-alcohol) was modeled as a within-person fixed effect. Initial models tested the main effect of stimulus type, evaluating whether alcohol cues elicited significantly different ERP responses compared to non-alcohol cues across the sample. Individual differences were examined by testing whether AUD severity moderated cue reactivity. Participants were dichotomized into “minimal” (no or mild AUD) and “pronounced” (moderate

or severe AUD) groups consistent with DSM-5 severity specifiers (mild: 2−3 symptoms; moderate: 4−5; severe: ≥6), and the primary test of severity differences in cue reactivity was the stimulus type × AUD group interaction, estimated using a mixed-effects model that included main effects and their interaction, with a random intercept for participant. When the stimulus type × AUD group interaction was significant, we decomposed the interaction by estimating the alcohol–non-alcohol contrast within each AUD group. To do so, we assessed the cue-type effect separately in the minimal group (AUD severity 0–1) and in the pronounced group (AUD severity 2–3), and computed simple-effects contrasts using estimated marginal means (EMMs). To evaluate whether findings in the minimal group were driven by inclusion of participants with no current AUD (0 criteria), we conducted a sensitivity analysis restricting the minimal group to mild-only (AUD severity = 1) and re-estimated the cue-type model. As an additional robustness check, we summarized within-person cue reactivity using a difference score ($\Delta P3b = P3b_{alcohol} - P3b_{non\text{-}alcohol}$) and compared ΔP3b between groups using a Welch independent-samples t-test, which is robust to heterogeneity of variance.

To test moderation by drinking behavior, retrospective self-report of typical alcohol consumption, objective transdermal ambulatory alcohol consumption, and self-report ambulatory alcohol consumption were each examined as moderators of cue-elicited ERP responses. Specifically, interaction terms between cue type (alcohol vs. non-alcohol) and each drinking index were included, allowing the magnitude of ERP reactivity to alcohol cues to be examined as a function of real-world drinking patterns. In these moderation models, each drinking index was treated as a between-person predictor and entered along with its interaction with stimulus type (stimulus type × moderator), estimated separately within each severity group (minimal;

pronounced). Significant stimulus type × moderator effects were probed with simple-slopes/simple-effects contrasts (i.e., estimating the alcohol–non-alcohol difference at representative values of the moderator) using planned linear contrasts. Alcohol use behavior–based moderators were tested in separate mixed-effects models (one moderator per model); moderators were not entered simultaneously due to conceptual overlap and multicollinearity among drinking indices. Alcohol use moderators were entered in their original measurement units (i.e., not standardized or transformed) to facilitate interpretation of effects in clinically meaningful terms. To support transparent inference, moderator results are reported as unstandardized fixed-effect estimates with 95% confidence intervals; for key stimulus type × moderator interactions, semi-partial R^2 values are also reported as an effect-size index.

3. Results

3.1 Participants

The mean total DSM-5 criteria count at screening was 3.55 (SD = 1.48; range = 2–8). Based on these scores, 36 participants (60%) were classified with “mild” AUD, 18 (30%) with “moderate” AUD, and 6 (10%) with “severe” AUD. When AUD severity was re-assessed using the full DSM-5 symptom checklists during the first laboratory visit (orientation session; see Procedures), the mean total DSM-5 criteria count was 3.73 (SD = 2.52; range: 0–11). Thirteen participants (21.7%) no longer met criteria for an AUD diagnosis. Among the remaining participants, 20 (33.3%) were classified as having a “mild” AUD, 16 (26.7%) as “moderate”, and 11 (18.3%) as “severe”. There was a moderate correlation between screening and laboratory symptom reports, $r = .581$, $p < .001$. Discrepancies between screening and session-based AUD-severity levels are likely attributable to a range of factors including: 1) Variation in symptom levels associated with the passage of time - an average of 14.05 days passed between screening

and participants' first laboratory session (*SD* = 10.23; range 1–42 days); 2) More exhaustive symptom assessment in the context of laboratory sessions; whereas a brief symptom checklist was incorporated into the screening questionnaire, full SCID-5 procedures were employed in the laboratory. In light of the more exhaustive and proximal assessment incorporated into in-person visits, analyses below incorporate DSM criteria assessed at laboratory sessions (see Table 1 for descriptive characteristics). Seven participants were not able to complete the Alcohol Image Viewing Task due to technical issues. In addition, six participants were excluded from ERP analyses due to excessive trial loss (fewer than 12 correct alcohol and/or non-alcohol target trials). The final analytic sample therefore comprised 47 participants: 11 (23.4%) with no AUD diagnosis, 16 (34.0%) with mild AUD, 12 (25.5%) with moderate AUD, and 8 (17.0%) with severe AUD. Given the obtained sample size (N=47) and observed variability, a sensitivity (minimum detectable effect) analysis (α=.05, two-tailed) indicated 80% power to detect a within-person alcohol–non-alcohol P3b difference of approximately 1.78 µV (dz≈0.42). For the severity comparison (n=27 minimal; n=20 pronounced), 80% power corresponded to detecting a between-group difference in cue-reactivity (ΔP3b) of approximately 3.42 µV (d≈0.85)

3.2 Overall Ambulatory Compliance and Alcohol Consumption

Rates of compliance with ambulatory assessment procedures were high. Participants responded to an average of 76.6% of prompts (*SD* = 18.08). All participants (100%) engaged in at least one drinking episode outside the laboratory over the 14-day ambulatory assessment period. On average, participants had at least one positive eBAC reading on 8.65 days (*SD* = 3.83), with an average eBAC during drinking days of 0.035% (*SD* = 0.011); with the highest participant-level mean peak reaching 0.092% (*SD* = 0.040).

On average, participants indicated "currently drinking" at least one time on 8.35 days (*SD* = 3.12), with an average of 5.08 drinks (*SD* = 2.49) per survey response submitted. See Table 2 for full descriptive statistics of eBAC and self-reports of ambulatory alcohol use assessment. eBAC corresponded closely with both daily self-reports of drinking and also with momentary drinking data. The correlation between daily self-reports of the number of drinks and the daily average eBAC was moderate in magnitude, $r = .459$, $t = 3.93$, $p < .001$. With respect to the momentary drinking data, when participants indicated "yes" to the question "Are you currently drinking?", 65.66% of the time the eBAC also was positive for that same time point. When participants indicated "no" to the question "Are you currently drinking?", 85.43% of the time the eBAC was also zero for that same time point.

3.3 Ambulatory Measurement Effects and Reactivity

Most participants did not report substantial reactivity to the transdermal sensor and/or the ambulatory survey procedures. Four participants indicated that they drank more than usual because of the ambulatory procedures, and 5 participants indicated that they drank less than usual because of the ambulatory procedures. Five participants indicated that they were sometimes uncomfortable or irritated by the ambulatory procedures. The remainder indicated no discomfort or behavioral changes linked with the ambulatory assessment procedures.

3.4 Behavioral Performance on the Oddball Task

On average, participants correctly responded on 96.59% of trials. Response accuracy did not significantly differ across Stimulus Types, $t(46) = 1.27$, $p = .211$ (96.99% for alcohol beverage images, 96.19% for non-alcohol beverage images).

3.5 ERP Analyses

A robust oddball effect was observed for the P3b (Figure 2), such that targets elicited reliably greater P3b amplitudes (M = 6.92 µV, SD = 5.88) than standards (M = 1.82 µV, SD = 4.12), b = 5.81, SE = 0.71, t = 8.19, p < .001. The effect was maximal over posterior electrode sites, consistent with canonical P3b topography. Visually, P3b responses were larger to alcohol beverage targets than to non-alcohol beverage targets. Analyses focused on the responses to the two beverage target types. These revealed a significant main effect of stimulus type, b = 1.43, SE = 0.62, t = 3.21, p = .025, with larger responses to alcohol beverage images (M = 7.63 µV, SD = 5.21) compared to non-alcohol beverage images (M = 6.20 µV, SD = 6.46).

Further analyses were conducted separately for participants with minimal AUD severity (non-AUD and mild AUD combined) and those with pronounced AUD severity (moderate and severe AUD combined). This division was applied to examine whether individual differences in the clinical severity of alcohol problems influence neural sensitivity to alcohol cues, as prior research suggests that cue reactivity is more pronounced among individuals with more severe symptom profiles (Namkoong et al., 2004; Petit et al., 2015; Littel et al., 2013, Figure 3). Consistent with this hypothesis, a significant interaction between Stimulus Type and AUD severity emerged (b = 2.89, SE = 1.19, t = 2.43, p = .019), prompting follow-up simple-effects tests within each severity group. In the minimal AUD group, the effect of stimulus type on P3b amplitude was non-significant, b = 0.20, SE = 0.92, t = 0.22, p = .827. Results were unchanged when considering only the mild group (AUD severity=1, alcohol vs. non-alcohol contrast remained non-significant, p = .816; see Supplement Table S2). In contrast, in the pronounced group, alcohol cues elicited significantly larger P3b amplitudes compared to non-alcohol cues, b = 3.10, SE = 0.61, t = 5.07, p < .001 (Figure 4). To probe the significant interaction, comparisons of EMMs tested the alcohol–non-alcohol contrast within each severity group. The alcohol–non-

alcohol contrast was non-significant in the minimal group (Δ = 0.203 μV, *SE* = 0.778, *t* = 0.260, *p* = .796) but significant in the pronounced group (Δ = 3.096 μV, *SE* = 0.904, *t* = 3.424, *p* = .001). As a robustness check, we also computed the participant-level difference score ($\Delta P3b = P3b_{alcohol} - P3b_{non\text{-}alcohol}$) and compared ΔP3b across groups. ΔP3b differed significantly between groups (independent-samples t-test *p* = .012), indicating larger cue-reactivity in the pronounced group than the minimal group.

Given prior work suggesting that P3b amplitudes are generally reduced in individuals with AUD, we also directly compared P3b amplitudes to the non-alcohol beverage targets across groups. P3b amplitudes to non-alcohol targets were larger ($t(44.94) = 2.99$, *p* = .004) in the minimal (*M* = 8.37 μV) compared to the pronounced (*M* = 3.27 μV) groups.

3.6 Moderators of Alcohol-Cue P3b

We examined whether real-world drinking, indexed by objective eBAC and self-report, moderated alcohol-cue P3b responses, and whether effects differed by severity group. For objective transdermal ambulatory alcohol use, no significant moderators emerged in the minimal group. By contrast, in the pronounced group, significant interactions indicated a tendency for alcohol-cue reactivity to scale with real-world drinking behavior. Specifically, among those with pronounced AUD, eBAC binge drinking days moderated the effect of stimulus type, *b* = 0.51, *SE* = 0.20, *t* = 2.55, *p* = .020, 95% CI [0.12, 0.90], semi-partial R^2= .27, with stronger alcohol-cue P3b responses associated with more binge days (Figure 5).

A similar effect was found for eBAC peak, *b* = 34.09, *SE* = 15.74, *t* = 2.17, *p* = .044, 95% CI [3.24, 64.94], semi-partial R^2= .21, indicating that, among participants with pronounced AUD, higher peak eBAC values correlated with greater alcohol-specific ERP amplitudes (Figure 6).

No other moderators, including additional eBAC indices, ambulatory self-reported measures, and retrospective reports, were found to significantly predict alcohol cue reactivity in either group. Full tables reporting all results of models can be found in the supplemental material.

3. Discussion

The current study used a combined laboratory–ambulatory design to examine whether neural markers of incentive salience for alcohol cues, indexed by the P3b, relate to real-world drinking behavior and whether these associations vary as a function of alcohol use disorder (AUD) severity. Consistent with predictions derived from incentive-salience models of addiction and the AARDoC framework, alcohol beverage images elicited larger P3b amplitudes than non-alcohol beverage images across the sample. Critically, however, this effect was not uniform: alcohol-specific P3b enhancement was concentrated among individuals with more pronounced AUD symptomatology, whereas individuals with minimal AUD showed comparable neural responses to alcohol and non-alcohol cues. These results highlight the importance of considering severity distinctions in AUD, as only those with pronounced symptoms demonstrated robust neurocognitive sensitivity to alcohol-related stimuli.

Consistent with this pattern, Kohen et al. (2024) showed that larger alcohol-cue–elicited P3b amplitudes prospectively track real-world consumption dynamics (e.g., steeper eBAC "front-loading" and elevated momentary craving) over multi-week EMA. Our results complement these findings by indicating that the expression of alcohol-cue P3b reactivity is not uniform but contingent on clinical severity: robust cue-elicited P3b differences emerged specifically among participants with moderate-to-severe AUD, whereas minimal-severity participants showed no reliable differentiation. However, because craving was not measured in

the present study, these findings should be interpreted as severity-linked differences in neural processing of alcohol versus non-alcohol cues rather than as direct evidence of craving-related mechanisms. In line with prior findings that alcohol-cue P3b indexes incentive salience relevant to naturalistic drinking, here we found that only biosensor-derived eBAC measures (and not self-reported drinking) moderated cue-elicited P3b, indicating that the translational utility of P3b is greatest where AUD severity is higher and objective exposure is elevated.

In the present data, the alcohol–non-alcohol cue salience contrast appears to reflect, in part, *attenuated* P3b responses to non-alcohol cues in the pronounced AUD group (rather than a selectively amplified absolute P3b response to alcohol cues). This interpretation is consistent with evidence that reduced P3/P3b amplitude is a robust feature of substance use disorders and AUD risk, and that smaller P3b amplitudes index broader disinhibitory/externalizing vulnerability that is associated with risk-taking and later substance-related problems (Euser et al., 2012; Patrick et al., 2006). Within this framework, one can see that alcohol cues are relatively prioritized because they elicit neural responses that are more comparable to normative responding, while responses to other cues remain blunted, consistent with the idea that alcohol-related stimuli may be among the few categories that reliably engage attentional/motivational systems in more severe AUD.

Interpretation of these findings should also consider that the present sample was relatively young (M age = 22.62 years), and the overall AUD symptom burden was modest (mean DSM-5 criteria approximately 3-4). National epidemiologic data indicate that AUD prevalence is highest among younger adults and that drinking patterns and symptoms can be developmentally dynamic during this period (Grant et al., 2015; National Institute on Alcohol Abuse and Alcoholism [NIAAA], 2025). Accordingly, the current results may be most

representative of earlier-stage AUD presentations with shorter lifetime drinking histories, rather than chronic decades-long exposure. Generalization to older or treatment-seeking samples with long-term heavy drinking should therefore be made cautiously, and future work should test whether severity-dependent alcohol-cue P3b patterns and their coupling with objective drinking indices replicate across a broader age range and illness chronicity (Koob & Volkow, 2016; Witkiewitz et al., 2019).

Beyond severity, our findings suggest that real-world drinking behaviors are associated with alcohol cue reactivity. In the pronounced AUD group, objective drinking indices moderated the stimulus effect. Individuals with stronger alcohol-cue P3b displayed more frequent binge-drinking and higher peak eBAC, whereas self-reports showed no moderation. In contrast, within the minimal group, drinking behaviors did not show any specific effect. Although daily self-reported number of drinks was moderately correlated with daily average eBAC, this correspondence did not translate into self-report moderation of cue-elicited P3b, suggesting that objective eBAC, rather than recalled quantity, may better capture the exposure dynamics that tune neural prioritization of alcohol cues. This pattern aligns with prior evidence that individual and contextual factors modulate cue salience and suggests that heavier naturalistic use potentiates neural prioritization of alcohol cues primarily when accompanied by higher clinical severity (Cofresí et al., 2019).

Importantly, these results suggest the potential incremental value of ambulatory alcohol assessment for linking drinking behavior to neurocognitive markers of addiction. Transdermal biosensor data were predictive of cue reactivity, whereas ambulatory and retrospective self-reports did not yield significant moderations. This finding aligns with concerns that retrospective recall of alcohol use is prone to biases and inaccuracies (Del Boca & Darkes, 2003; Stockwell et

al., 2004). By contrast, biosensor data can capture intoxication levels as they occur in daily life, thereby reducing memory-related distortions and potentially offering a more ecologically valid index of consumption. Ambulatory self-reports, while valuable, may be influenced by limitations such as impaired awareness of intake during intoxication (Weissenborn & Duka, 2003; White, 2003), variability in standard drink definitions (Barnett et al., 2009; Kerr et al., 2008), and social desirability biases (Davis et al., 2010; Zapolski et al., 2014). Taken together, these findings indicate that technology-mediated ambulatory approaches may provide a powerful complement to traditional methods, potentially enabling researchers to more accurately capture drinking behaviors as they unfold in the natural environment.

A further contribution of this study is showing that neurocognitive responses to alcohol cues are associated with both ongoing drinking patterns and clinically defined AUD severity. Enhanced alcohol cue reactivity was observed exclusively among those with moderate-to-severe AUD, consistent with prior work showing greater P3b responses to alcohol cues in clinical samples compared to social drinkers (Namkoong et al., 2004; Littel et al., 2013; Petit et al., 2015). Most previous studies, however, were conducted prior to the adoption of DSM-5 AUD severity specifiers and thus did not differentiate between mild and more severe presentations. Our results suggest that individuals with moderate-to-severe AUD display distinctive neurocognitive signatures relative to those with minimal AUD, aligning with broader findings in psychiatry that symptom severity can mark qualitatively different clinical and functional profiles (Regier et al., 2013).

4.1 Limitations

Despite several strengths, the present study involved constraints that temper inference. The sample size, while comparable to prior ambulatory EEG/EMA investigations, was modest;

replication with a larger cohort is warranted to stabilize effect-size estimates and enable finer-grained modeling. The two-week ambulatory window captured substantial variability in everyday use for most participants, yet longer follow-ups would better characterize stability and change in brain–behavior coupling. Measurement considerations also apply. Craving was not assessed in this dataset. We did not administer a validated clinical craving measure (nor collect momentary craving ratings), which limits our ability to determine whether severity-related P3b differences reflect AUD severity per se versus severity-related craving or other correlated processes. Future work should include standardized craving assessments (clinical and/or EMA) to directly evaluate craving as a mediator or moderator of alcohol-cue–elicited neural responses.

Additionally, the alcohol cues in the oddball task were generic beverage photographs rather than participant-preferred (idiographic) alcohol cues. Because beverage preference can influence cue-elicited ERP responses (Thurin et al., 2017), this may have introduced heterogeneity in cue relevance that dampened cue effects for some participants. In the present study, pronounced AUD participants showed robust P3b differentiation even with generic cues, suggesting generalized incentive salience for alcohol stimuli; however, any mismatch between pictured beverages and personal preferences would be expected to attenuate effect sizes and could contribute to null effects in lower-severity participants. Future studies could (i) collect preferred beverage type and cue ratings (e.g., familiarity, liking, craving), (ii) use preference-matched cue sets (beer vs wine vs liquor) or fully idiographic cues (Kirsch et al., 2025; Amlung et al., 2024), and (iii) balance personalization with standardization by drawing from validated alcohol picture libraries while matching to participant preference (Stauffer et al., 2017).

Although eBAC values derived from transdermal sensing correlate with drinking-quantity estimates, they are vulnerable to person-level and contextual confounds (e.g., skin

properties, perfusion, device placement, ambient temperature), making them biased and imprecise as point estimates of BAC; convergent validation with breathalyzer sampling and alcohol-administration paradigms is therefore essential to bolster construct validity. Ambulatory survey compliance was lower among heavier drinkers, raising the possibility of state-dependent missingness; future work should consider briefer prompts, adaptive scheduling, and incentive structures to improve response rates. Causal interpretation remains limited by the cross-context bridge (lab ERP to field behavior); repeated neural assessments or cross-lagged/time-varying models would help address directionality. Analytically, a higher-order interaction framework, as testing a three-way interaction in which the neural index of cue salience varies as a function of individual severity and drinking patterns, would have provided a more incisive test of conditional dependencies; however, the available sample and event counts did not provide sufficient power for such models. Relatedly, multiple alcohol-use indices were evaluated as moderators in planned, conceptually grouped models. Although these indices are correlated and the analyses were hypothesis-driven, testing multiple moderators can still increase the chance of Type I error. Accordingly, moderator findings, especially those observed in the pronounced AUD subgroup, should be viewed as provisional and in need of replication. Future studies with larger samples will be critical for testing fully dimensional moderation models in which AUD symptom count, cue type, and real-world drinking jointly interact. Finally, comprehensive assessment of substance use and alcohol–substance co-use was beyond the scope of the present study. Future research should incorporate systematic measurement of cannabis and other substance use, particularly given increasing population-level prevalence, to clarify how co-use may shape alcohol cue reactivity and brain–behavior associations.

4. Conclusion

In summary, P3b alcohol cue salience appears shaped by both AUD severity and drinking patterns: individuals with pronounced AUD show heightened neural responses to alcohol cues, and these responses are further amplified among those engaging in more frequent/heavier use (Kohen et al., 2024; Fairbairn & Bosch, 2021). These findings support viewing P3b as a severity-sensitive, ecologically relevant marker within incentive-salience accounts of addiction (Robinson & Berridge, 1993; Koob & Volkow, 2016) and underscore the value of integrating DSM-5 severity specifiers with biosensor-based, real-world measurement when examining mechanisms and designing interventions that target cue reactivity in those most vulnerable.

Disclosures and acknowledgments

This research was supported by NIH grants R01AA025969 and R01AA028488 awarded to Catharine Fairbairn, and grants F31AA028990 and K01AA032018 to Dahyeon Kang. We extend our sincere thanks to Talia Ariss, Nigel Bosch, and the students and staff of the Alcohol Research Laboratory at the University of Illinois at Urbana-Champaign for their invaluable assistance in conducting this research.

References

Alcohol and young adults ages 18 to 25. National Institutes of Health. Retrieved December 18, 2025, from American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). https://doi.org/10.1176/appi.books.9780890425596

Amlung, M., Marsden, E., Hargreaves, T., Sweet, L. H., Murphy, J. G., & Mackillop, J. (2024). Neural correlates of increased alcohol demand following alcohol cue exposure in adult heavy drinkers. *Psychiatry Research: Neuroimaging*, *340*, 111809. https://doi.org/10.1016/j.pscychresns.2024.111809

Ariss, T., Caumiant, E. P., Fairbairn, C. E., Kang, D., Bosch, N., & Morris, J. K. (2025). Exploring associations between drinking contexts and alcohol consumption: An analysis of photographs. *Journal of Psychopathology and Clinical Science.* https://doi.org/10.1037/abn0000977

Ariss, T., Fairbairn, C. E., & Bosch, N. (2023). Examining new-generation transdermal alcohol biosensor performance across laboratory and field contexts. *Alcohol: Clinical and Experimental Research, 47*(1), 50–59. https://doi.org/10.1111/acer.14977

Armor, D. J., Polich, J. M., & Stambul, H. B. (1978). Reliability and validity of self-reported drinking behavior. *Alcoholism and Treatment,* 173–211.

Barnett, N. P., Wei, J., & Czachowski, C. (2009). Measured alcohol content in college party mixed drinks. *Psychology of Addictive Behaviors, 23*(1), 152–156. https://doi.org/10.1037/a0013611

Bartholow, B. D., Henry, E. A., & Lust, S. A. (2007). Effects of alcohol sensitivity on P3 event-related potential reactivity to alcohol cues. *Psychology of Addictive Behaviors, 21*(4), 555–563. https://doi.org/10.1037/0893-164X.21.4.555

Bartholow, B. D., Lust, S. A., & Tragesser, S. L. (2010). Specificity of P3 event-related potential reactivity to alcohol cues in individuals low in alcohol sensitivity. *Psychology of Addictive Behaviors, 24*(2), 220–228. https://doi.org/10.1037/a0017705

Bates, D., Mächler, M., Bolker, B., & Walker, S. (2015). Fitting linear mixed-effects models using lme4. *Journal of Statistical Software, 67*(1), 1–48. https://doi.org/10.18637/jss.v067.i01

Carter, B. L., & Tiffany, S. T. (1999). Meta-analysis of cue-reactivity in addiction research. *Addiction, 94*(3), 327–340. https://doi.org/10.1046/j.1360-0443.1999.9433273.x

Cofresí, R. U., Bartholow, B. D., & Piasecki, T. M. (2019). Evidence for incentive salience sensitization as a pathway to alcohol use disorder. *Neuroscience & Biobehavioral Reviews, 107,* 897–926. https://doi.org/10.1016/j.neubiorev.2019.10.009

Cofresí, R. U., Kohen, C. B., Motschman, C. A., Wiers, R. W., Piasecki, T. M., & Bartholow, B. D. (2022). Behavioral response bias and event-related brain potentials implicate elevated incentive salience attribution to alcohol cues in emerging adults with lower sensitivity to alcohol. *Addiction, 117*(4), 892–904. https://doi.org/10.1111/add.15728

Cox, W. M., Schippers, G. M., Klinger, E., Skutle, A., Stuchlíková, I., Man, F., King, A. L., & Inderhaug, R. (2002). Motivational structure and alcohol use of university students across four nations. *Journal of Studies on Alcohol, 63*(3), 280–285. https://doi.org/10.15288/jsa.2002.63.280

Davis, C. G., Thake, J., & Vilhena, N. (2010). Social desirability biases in self-reported alcohol consumption and harms. *Addictive Behaviors, 35*(4), 302–311. https://doi.org/10.1016/j.addbeh.2009.11.001

Del Boca, F. K., & Darkes, J. (2003). The validity of self-reports of alcohol consumption: State of the science and challenges for research. *Addiction, 98*(s2), 1–12. https://doi.org/10.1046/j.1359-6357.2003.00586.x

Didier, N. A., King, A. C., Polley, E. C., & Fridberg, D. J. (2024). Signal processing and machine learning with transdermal alcohol concentration to predict natural environment alcohol consumption. *Experimental and Clinical Psychopharmacology, 32*(2), 245. https://doi.org/10.1037/pha0000683

Donchin, E. (1981). Surprise!… surprise?. *Psychophysiology*, *18*(5), 493-513. https://doi.org/10.1111/j.1469-8986.1981.tb01815.x

Ehlers, C. L., Phillips, E., Sweeny, A., & Slawecki, C. J. (2003). Event-related potential responses to alcohol-related stimuli in African–American young adults: Relation to family history of alcoholism and drug usage. *Alcohol and Alcoholism, 38*(4), 332–338. https://doi.org/10.1093/alcalc/agg080

Euser, A. S., Arends, L. R., Evans, B. E., Greaves-Lord, K., Huizink, A. C., & Franken, I. H. (2012). The P300 event-related brain potential as a neurobiological endophenotype for

substance use disorders: a meta-analytic investigation. *Neuroscience & Biobehavioral Reviews*, *36*(1), 572-603. https://doi.org/10.1016/j.neubiorev.2011.09.002

Fairbairn, C. E., & Bosch, N. (2021). A new generation of transdermal alcohol biosensing technology: Practical applications, machine learning analytics, and questions for future research. *Addiction, 116*(10), 2912–2920. https://doi.org/10.1111/add.15523

Fairbairn, C. E., & Kang, D. (2020). Transdermal alcohol monitors: Research, applications, and future directions. In D. Frings & I. Albery (Eds.), *The Handbook of Alcohol Use and Abuse.* Elsevier. https://doi.org/10.1016/B978-0-12-816720-5.00014-1

Fairbairn, C. E., Bresin, K., Kang, D., Rosen, I. G., Ariss, T., Luczak, S. E., Barnett, N. P., & Eckland, N. S. (2018). A multimodal investigation of contextual effects on alcohol's emotional rewards. *Journal of Abnormal Psychology, 127*(4), 359–373. https://doi.org/10.1037/abn0000346

Fairbairn, C. E., Kang, D., & Bosch, N. (2020). Using machine learning for real-time BAC estimation from a new-generation transdermal biosensor in the laboratory. *Drug and Alcohol Dependence, 216,* 108205. https://doi.org/10.1016/j.drugalcdep.2020.108205

Fairbairn, C. E., Kang, D., & Federmeier, K. D. (2021). Alcohol and neural dynamics: A meta-analysis of acute alcohol effects on event-related brain potentials. *Biological Psychiatry.* https://doi.org/10.1016/j.biopsych.2020.11.024

First, M. B., Williams, J. B. W., Karg, R. S., & Spitzer, R. L. (2015). *Structured clinical interview for DSM-5®—Research version (SCID-5-RV, Version 1.0.0).* American Psychiatric Association.

Genkina, O. A., & Shostakovich, G. S. (1983). Elaboration of a conditioned reflex in chronic alcoholics using an unrecognizable motivationally significant word. *Zhurnal Vysshei Nervnoi Deiatelnosti Imeni I. P. Pavlova, 33*(6), 1010–1018.

Grant, B. F., Goldstein, R. B., Saha, T. D., Chou, S. P., Jung, J., Zhang, H., Pickering, R. P., Ruan, W. J., Smith, S. M., Huang, B., & Hasin, D. S. (2015). Epidemiology of DSM-5 alcohol use disorder: results from the National Epidemiologic Survey on Alcohol and Related Conditions III. *JAMA psychiatry*, *72*(8), 757-766. https://doi.org/10.1001/jamapsychiatry.2015.0584

Hasin, D. S., O'Brien, C. P., Auriacombe, M., Borges, G., Bucholz, K., Budney, A., Compton W. M., Crowley T., Ling W., Petry N. M., Schuckit M., & Grant, B. F. (2013). DSM-5

criteria for substance use disorders: recommendations and rationale. *American Journal of Psychiatry, 170*(8), 834-851. https://doi.org/10.1176/appi.ajp.2013.1206078

Herrmann, M. J., Weijers, H. G., Wiesbeck, G. A., Aranda, D., Böning, J., & Fallgatter, A. J. (2000). Event-related potentials and cue-reactivity in alcoholism. *Alcoholism: Clinical and Experimental Research, 24*(11), 1724–1739. https://doi.org/10.1111/j.1530-0277.2000.tb01974.x

Herrmann, M. J., Weijers, H. G., Wiesbeck, G. A., Böning, J., & Fallgatter, A. J. (2001). Alcohol cue-reactivity in heavy and light social drinkers as revealed by event-related potentials. *Alcohol and Alcoholism, 36*(6), 588–593. https://doi.org/10.1093/alcalc/36.6.588

https://www.niaaa.nih.gov/alcohols-effects-health/alcohol-topics-z/alcohol-facts-and-statistics/alcohol-and-young-adults-ages-18-25

Jasper, H. H. (1958). Ten-twenty electrode system of the international federation. *Electroencephalogr Clin Neurophysiol*, *10*, 371-375.

Kambouropoulos, N., & Staiger, P. K. (2009). 'Cue reward salience' predicts craving in response to alcohol cues. *Personality and Individual Differences, 46*(2), 78–82. https://doi.org/10.1016/j.paid.2008.08.023

Kenny, D. A., Kashy, D. A., & Cook, W. L. (2020). *Dyadic data analysis*. Guilford Publications.

Kerr, W. C., Patterson, D., Koenen, M. A., & Greenfield, T. K. (2008). Alcohol content variation of bar and restaurant drinks in Northern California. *Alcoholism: Clinical and Experimental Research, 32*(9), 1623–1629. https://doi.org/10.1111/j.1530-0277.2008.00741.x

Kiluk, B. D., Ray, L. A., Walthers, J., Bernstein, M., Tonigan, J. S., & Magill, M. (2019). Technology-delivered cognitive-behavioral interventions for alcohol use: a meta-analysis. *Alcoholism: clinical and experimental research, 43(*11), 2285-2295. https://doi.org/10.1111/acer.14189

Kirsch, D. E., Grodin, E. N., Leggio, L., McKee, S. A., Meredith, L. R., Mereish, E. H., ... & Ray, L. A. (2025). The alcohol cue-exposure paradigm as a screening tool for alcohol use disorder medication development: A critical review. *Alcohol: Clinical and Experimental Research*. https://doi.org/10.1111/acer.70170

Kohen, C. B., Cofresí, R. U., Piasecki, T. M., & Bartholow, B. D. (2024). Predictive utility of the P3 event-related potential (ERP) response to alcohol cues for ecologically assessed alcohol craving and use. *Addiction Biology, 29*(2), e13368. https://doi.org/10.1111/adb.13368

Koob, G. F., & Volkow, N. D. (2016). Neurobiology of addiction: A neurocircuitry analysis. *The Lancet Psychiatry, 3*(8), 760–773. https://doi.org/10.1016/S2215-0366(16)00104-8

Krank, M. D., & Wall, A. M. (2006). Context and retrieval effects on implicit cognitions for substance use. In *Handbook of Implicit Cognition and Addiction* (pp. 281–292). SAGE Publications, Inc. https://doi.org/10.4135/9781412976237.n19

Kuznetsova, A., Brockhoff, P. B., & Christensen, R. H. B. (2017). lmerTest Package: Tests in Linear Mixed Effects Models. *Journal of Statistical Software*, *82*(13), 1–26. https://doi.org/10.18637/jss.v082.i13

Kwako, L. E., Momenan, R., Litten, R. Z., Koob, G. F., & Goldman, D. (2016). Addictions neuroclinical assessment: a neuroscience-based framework for addictive disorders. *Biological psychiatry*, *80*(3), 179-189. https://doi.org/10.1016/j.biopsych.2015.10.024

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (2008). *International Affective Picture System (IAPS): Affective ratings of pictures and instruction manual.* Technical Report A-8.

Littel, M., Field, M., van de Wetering, B. J. M., & Franken, I. H. A. (2013). Reduced cognitive processing of alcohol cues in alcohol-dependent patients seeking treatment: An ERP study. *Journal of Experimental Psychopathology, 4*(3), 291–302. https://doi.org/10.5127/jep.027412

Litten, R. Z., Ryan, M. L., Falk, D. E., Reilly, M., Fertig, J. B., & Koob, G. F. (2015). Heterogeneity of alcohol use disorder: understanding mechanisms to advance personalized treatment. *Alcoholism, clinical and experimental research*, *39*(4), 579-584. https://doi.org/10.1111/acer.12669

MacKillop, J., & Lisman, S. A. (2008). Effects of a context shift and multiple context extinction on reactivity to alcohol cues. *Experimental and Clinical Psychopharmacology, 16*(4), 322–331. https://doi.org/10.1037/a0012686

Martins, J. S., Bartholow, B. D., Cooper, M. L., Irvin, K. M., & Piasecki, T. M. (2019). Interactive effects of naturalistic drinking context and alcohol sensitivity on neural alcohol cue-reactivity responses. *Alcoholism: Clinical and Experimental Research, 43*(8), 1777–1789. https://doi.org/10.1111/acer.14134

Mellentin, A. I., Cox, W. M., Fadardi, J. S., Martinussen, L., Mistarz, N., Skøt, L., Rømer T. K. , Mathiasen K. , Lichtenstein M., & Nielsen, A. S. (2021). A randomized controlled trial of

attentional control training for treating alcohol use disorder. Frontiers in Psychiatry, 12, 748848. https://doi.org/10.3389/fpsyt.2021.748848

Namkoong, K., Lee, E., Lee, C. H., Lee, B. O., & An, S. K. (2004). Increased P3 amplitudes induced by alcohol-related pictures in patients with alcohol dependence. *Alcoholism: Clinical and Experimental Research, 28*(9), 1317–1323. https://doi.org/10.1097/01.ALC.0000139828.78099.69

National Institute on Alcohol Abuse and Alcoholism. (2004). *NIAAA council approves definition of binge drinking.* NIAAA Newsletter. https://www.niaaa.nih.gov/sites/default/files/newsletters/Newsletter_Number3.pdf

National Institute on Alcohol Abuse and Alcoholism. (2025, August 12).

National Institute on Alcohol Abuse and Alcoholism. (2018). *Recommended Alcohol Questions*. https://www.niaaa.nih.gov/research/guidelines-and-resources/recommended-alcohol-questions

Niaura, R., Wilson, G. T., & Westrick, E. (1988). Self-awareness, alcohol consumption, and reduced cardiovascular reactivity. *Psychosomatic Medicine, 50*(4), 360–380. https://doi.org/10.1097/00006842-198807000-00005

Patrick, C. J., Bernat, E. M., Malone, S. M., Iacono, W. G., Krueger, R. F., & McGue, M. (2006). P300 amplitude as an indicator of externalizing in adolescent males. *Psychophysiology*, *43*(1), 84-92. https://doi.org/10.1111/j.1469-8986.2006.00376.x

Paulus, M. P., Tapert, S. F., & Schulteis, G. (2009). The role of interoception and alliesthesia in addiction. *Pharmacology Biochemistry and Behavior*, *94*(1), 1-7. https://doi.org/10.1016/j.pbb.2009.08.005

Petit, G., Cimochowska, A., Cevallos, C., Cheron, G., Kornreich, C., Hanak, C., Schroder E., Verbanck P., & Campanella, S. (2015). Reduced processing of alcohol cues predicts abstinence in recently detoxified alcoholic patients in a three-month follow up period: An ERP study. *Behavioural Brain Research, 282,* 84–94. https://doi.org/10.1016/j.bbr.2014.12.057

Petit, G., Kornreich, C., Dan, B., Verbanck, P., & Campanella, S. (2014). Electrophysiological correlates of alcohol- and non-alcohol-related stimuli processing in binge drinkers: A follow-up study. *Journal of Psychopharmacology, 28*(11), 1041–1052. https://doi.org/10.1177/0269881114545663

Petit, G., Kornreich, C., Verbanck, P., & Campanella, S. (2013). Gender differences in reactivity to alcohol cues in binge drinkers: A preliminary assessment of event-related potentials. *Psychiatry Research, 209*(3), 494–503. https://doi.org/10.1016/j.psychres.2013.04.005

Polich, J. (2007). Updating P300: An integrative theory of P3a and P3b. *Clinical Neurophysiology, 118*(10), 2128–2148. https://doi.org/10.1016/j.clinph.2007.04.019

Polich, J., & Kok, A. (1995). Cognitive and biological determinants of P300: An integrative review. *Biological Psychology, 41*(2), 103–146. https://doi.org/10.1016/0301-0511(95)05130-9

Porjesz, B., Rangaswamy, M., Kamarajan, C., Jones, K. A., Padmanabhapillai, A., & Begleiter, H. (2005). The utility of neurophysiological markers in the study of alcoholism. *Clinical Neurophysiology, 116*(5), 993–1018. https://doi.org/10.1016/j.clinph.2004.12.016

Pulido, C., Brown, S. A., Cummins, K., Paulus, M. P., & Tapert, S. F. (2010). Alcohol cue reactivity task development. *Addictive behaviors*, *35*(2), 84-90. https://doi.org/10.1016/j.addbeh.2009.09.006

Regier, D. A., Kuhl, E. A., & Kupfer, D. J. (2013). The DSM-5: Classification and criteria changes. *World Psychiatry, 12*(2), 92–98. https://doi.org/10.1002/wps.20050

Robinson, Michael J. F., Terry E. Robinson, and Kent C. Berridge, 'Incentive Salience in Addiction and Over-Consumption', in Stephanie D. Preston, Morten L. Kringelbach, and Brian Knutson (eds), *The Interdisciplinary Science of Consumption* (Cambridge, MA, 2014; online edn, MIT Press Scholarship Online, 22 Jan. 2015), https://doi.org/10.7551/mitpress/9780262027670.003.0010, accessed 30 Oct. 2025.

Robinson, T. E., & Berridge, K. C. (1993). The neural basis of drug craving: an incentive-sensitization theory of addiction. *Brain research reviews*, *18*(3), 247-291. https://doi.org/10.1016/0165-0173(93)90013-P

Shiffman, S., Stone, A. A., & Hufford, M. R. (2008). Ecological momentary assessment. *Annual Review of Clinical Psychology, 4*, 1–32. https://doi.org/10.1146/annurev.clinpsy.3.022806.091415

Stauffer, C. S., Dobberteen, L., & Woolley, J. D. (2017). American Alcohol Photo Stimuli (AAPS): A standardized set of alcohol and matched non-alcohol images. *The American Journal of Drug and Alcohol Abuse*, *43*(6), 647-655. https://doi.org/10.1080/00952990.2016.1253093

Stockwell, T., Donath, S., Cooper-Stanbury, M., Chikritzhs, T., Catalano, P., & Mateo, C. (2004). Under-reporting of alcohol consumption in household surveys: A comparison of quantity–frequency, graduated–frequency and recent recall. *Addiction, 99*(8), 1024–1033. https://doi.org/10.1111/j.1360-0443.2004.00815.x

Stone, A. A., & Shiffman, S. (1994). Ecological momentary assessment (EMA) in behavioral medicine. *Annals of Behavioral Medicine, 16*(3), 199–202. https://doi.org/10.1093/abm/16.3.199

Stormark, K. M., Field, N. P., Hugdahl, K., & Horowitz, M. (1997). Selective processing of visual alcohol cues in abstinent alcoholics: An approach-avoidance conflict? *Addictive Behaviors, 22*(4), 509–519. https://doi.org/10.1016/S0306-4603(96)00051-2

Stritzke, W. G. K., Breiner, M. J., Curtin, J. J., & Lang, A. R. (2004). Assessment of substance cue reactivity: Advances in reliability, specificity, and validity. *Psychology of Addictive Behaviors, 18*(2), 148–159. https://doi.org/10.1037/0893-164X.18.2.148

Thurin, K., Ceballos, N. A., & Graham, R. (2017). Alcohol preferences and event-related potentials to alcohol images in college students. *Journal of studies on alcohol and drugs*, *78*(6), 916-921. https://doi.org/10.15288/jsad.2017.78.916

Valyear, M. D., Villaruel, F. R., & Chaudhri, N. (2017). Alcohol-seeking and relapse: A focus on incentive salience and contextual conditioning. *Behavioural Processes, 141,* 26–32. https://doi.org/10.1016/j.beproc.2017.04.019

Villaruel, F. R., & Chaudhri, N. (2016). Individual differences in the attribution of incentive salience to a Pavlovian alcohol cue. *Frontiers in Behavioral Neuroscience, 10.* https://doi.org/10.3389/fnbeh.2016.00238

Weissenborn, R., & Duka, T. A. (2003). Acute alcohol effects on cognitive function in social drinkers: Their relationship to drinking habits. *Psychopharmacology, 165*(3), 306–312. https://doi.org/10.1007/s00213-002-1281-1

White, A. M. (2003). What happened? Alcohol, memory blackouts, and the brain. *Alcohol Research & Health, 27*(2), 186–196.

Witkiewitz, K., Litten, R. Z., & Leggio, L. (2019). Advances in the science and treatment of alcohol use disorder. *Science Advances, 5*(9), eaax4043. https://doi.org/10.1126/sciadv.aax4043

Zapolski, T. C. B., Pedersen, S. L., McCarthy, D. M., & Smith, G. T. (2014). Less drinking, yet more problems: Understanding African American drinking and related problems. *Psychological Bulletin, 140*(1), 188–223. https://doi.org/10.1037/a0032113

Table 1. Descriptive Characteristics of Sample

	All Participants (n=60)	No AUD (n=13)	Mild AUD (n=20)	Moderate AUD (n=16)	Severe AUD (n=11)
Gender					
n(%) Male	31(51.7)	6 (46.2)	11 (55.0)	10 (62.5)	4 (36.3)
n(%) Female	29 (48.3)	7 (53.8)	9 (45.0)	6 (37.5)	7 (63.6)
Race					
n(%) White	39 (65.0)	8 (61.5)	12 (60.0)	10 (62.5)	9 (81.8)
n(%) African American	5 (8.3)	0 (0)	2 (10.0)	1 (6.3)	2 (18.2)
n(%) Asian	13 (21.7)	5 (38.5)	4 (20.0)	4 (25.0)	0 (0)
n(%) American Indian or Alaska Native	1 (1.7)	0 (0)	1 (5.0)	0 (0)	0 (0)
n(%) Pacific Islander	1 (1.7)	0 (0)	0 (0)	1 (6.3)	0 (0)
n(%) Multi-racial	1 (1.7)	0 (0)	1 (5.0)	0 (0)	0 (0)
Ethnicity					
n(%) Hispanic	12 (20.0)	1 (7.7)	2 (10.0)	6 (37.5)	3 (27.3)
n(%) Not Hispanic	48 (80.0)	12 (92.3)	18 (90.0)	10 (62.5)	8 (72.7)
Age					
mean (SD)	22.62 (2.88)	21.85 (1.46)	22.10 (1.45)	22.56 (3.18)	24.55 (4.68)
median (IQR*)	21 (21-23)	21 (21-22)	22 (21-22)	21 (21-22)	22 (21-29)
Status					
n(%) full time student	52(85.7)	13(100)	18(90.0)	13(81.3)	8(72.7)
n(%) part time student	6(10.0)	0(0)	2(10.0)	2(12.5)	2(18.2)
n(%) not student	2(3.3)	0(0)	0(0)	1(6.3)	1(9.1)

*IQR: Interquartile Range.

Table 2. Descriptive Statistics for Alcohol Consumption Levels by AUD Severity for the complete sample

	All (n = 60)		No AUD (n = 13)		Mild AUD (n = 20)		Moderate AUD (n = 16)		Severe AUD (n = 11)	
	Mean (SD)	Range	Mean (SD)	Range	Mean (SD)	Range	Mean (SD)	Range	Mean (SD)	Range
Retrospective Self-Report										
Drinking Frequency	14.12 (5.59)	6-29	13.77 (4.68)	8-25	14.89 (6.33)	6-29	12.69 (4.85)	6-20	15.91 (6.28)	6-28
Drinking Quantity	5.65 (4.44)	2-25	3.38 (1.33)	2-6	5.67 (3.29)	2-15	6.06 (4.89)	2-20	7.82 (6.84)	3-25
Binge Drinking Frequency	8.17 (4.65)	1-20	5.23 (3.49)	1-13	9.00 (4.59)	3-20	7.31 (3.55)	2-15	11.91 (5.34)	5-20
Ambulatory eBAC										
Drinking Days	8.65 (3.83)	0-14	8.31 (3.17)	4-14	8.39 (3.88)	0-14	8.63 (4.19)	1-14	9.18 (4.60)	1-14
Drinking Frequency	0.74 (0.16)	0.00-0.95	0.79 (0.11)	0.60-0.92	0.71 (0.16)	0.29-0.94	0.79 (0.11)	0.56-0.95	0.66 (0.24)	0.00-0.87
Drinking Quantity	0.03 (0.01)	0.00-0.07	0.03 (0.01)	0.02-0.05	0.03 (0.01)	0.00-0.06	0.04 (0.01)	0.03-0.07	0.04 (0.01)	0.02-0.05
Peak Max	0.09 (0.04)	0.00 0.23	0.09 (0.03)	0.04 0.12	0.09 (0.04)	0.00 0.17	0.10 (0.05)	0.04 0.23	0.09 (0.04)	0.04 0.14
Binge Drinking Frequency	1.78 (2.20)	0-10	1.31 (1.38)	0-4	1.89 (2.11)	0-7	1.94 (2.77)	0-10	1.91 (2.47)	0-7
Ambulatory Self-Report										
Drinking Days	8.35 (3.12)	1-14	8.23 (3.44)	3-14	7.56 (3.17)	4-14	8.38 (3.34)	1-14	9.27 (2.28)	6-13
Drinking Frequency	0.22 (0.14)	0.00-0.71	0.19 (0.12)	0.00-0.47	0.20 (0.17)	0.00-0.71	0.21 (0.14)	0.00-0.50	0.29 (0.13)	0.08-0.58
Drinking Quantity	5.08 (2.49)	1.40-12.67	4.42 (2.90)	2.13-12.67	5.16 (2.45)	1.40-9.33	5.24 (1.99)	2.67-9.0	4.83 (2.39)	2.17-9.38
Binge Drinking Frequency	4.42 (2.74)	0-12	3.00 (1.78)	1-8	4.56 (3.01)	0-12	4.38 (2.47)	1-10	5.18 (3.03)	2-12

Self-Reported Peak day	5.43 (3.10)	1-15	3.62 (2.06)	1-9	6.20 (3.35)	1-13	6.19 (3.25)	2-15	5.09 (2.84)	3-13

Figure 1. Study timeline and data-collection sequence.

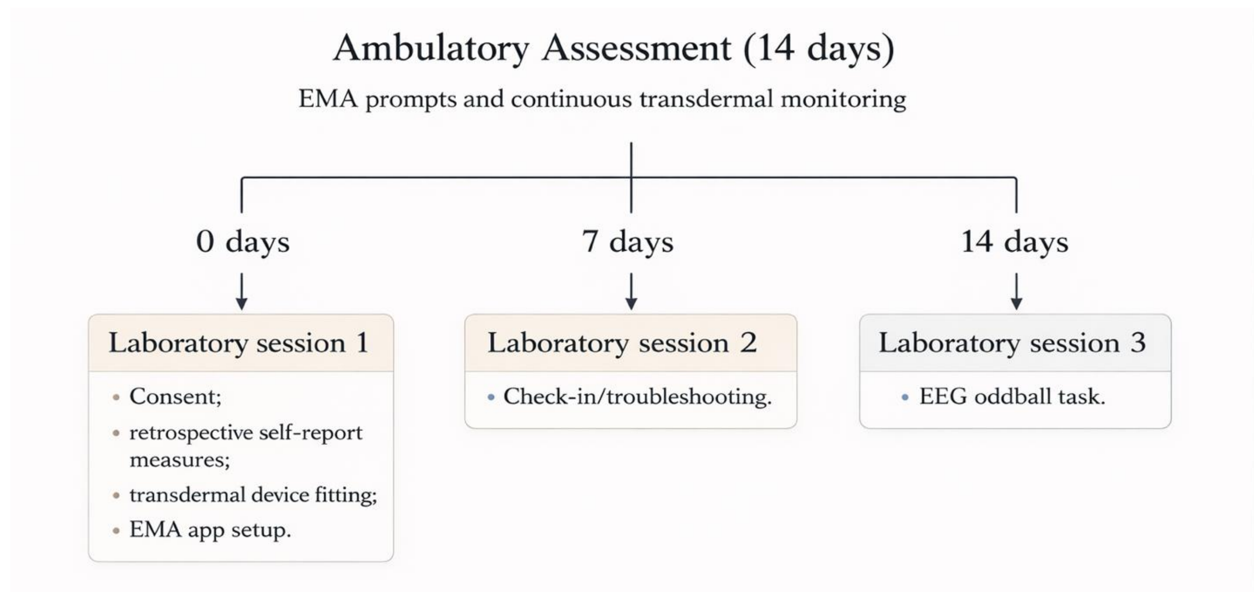
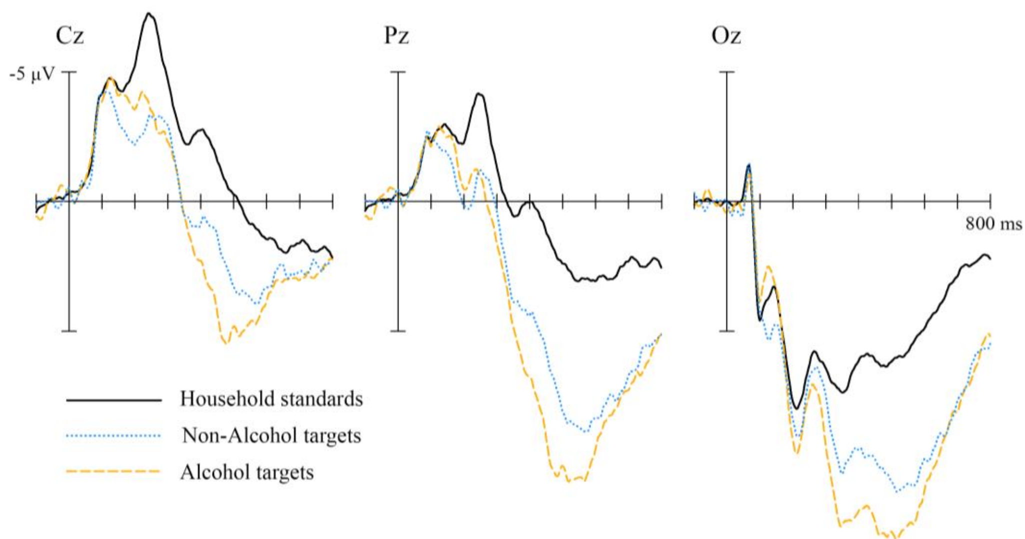
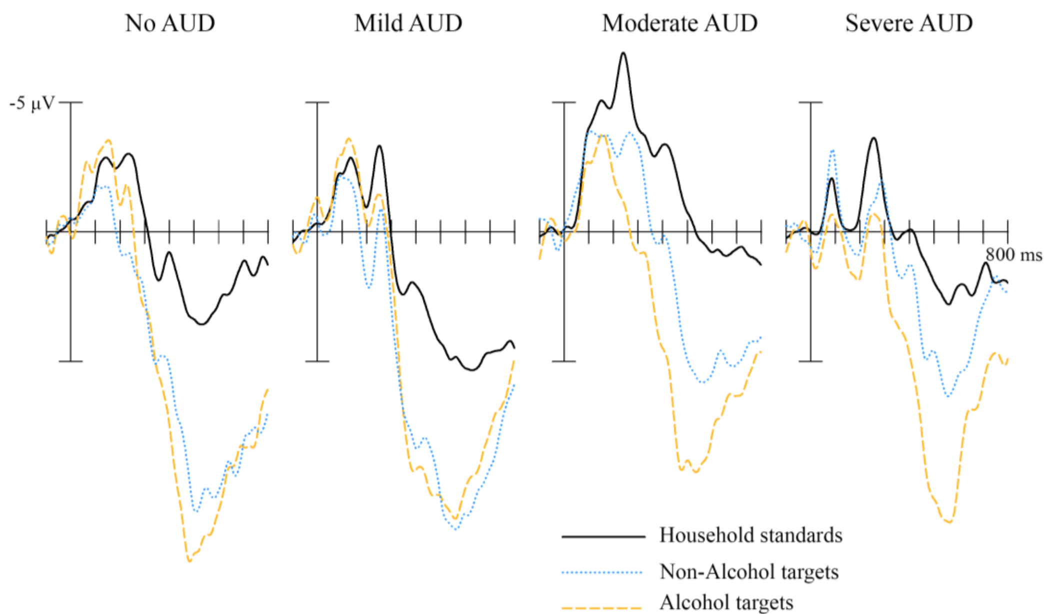


Figure 2. Event-related brain potential waveforms elicited by target alcohol, target non-alcohol, and standard household images.



Note: ERP Waveforms for standard (household) images (solid black), non-alcohol targets (green dotted), and alcohol targets (red dashed) are plotted at the three centro-posterior midline electrodes (Cz, Pz, and Oz) for all participants combined (N=47). Stimulus onset occurred at 0 ms. Negative voltage is plotted up.

Figure 3. Alcohol Cue Salience as a Function of AUD Severity



Note: ERP Waveforms for standard (household) images (solid black), non-alcohol targets (green dotted), and alcohol targets (red dashed) are plotted at channel Pz for participants with differing levels of AUD severity. Stimulus onset occurred at 0 ms. Negative voltage is plotted up. Alcohol cue salience (larger P3b responses to alcohol than to non-alcohol targets) is enhanced in participants with moderate to severe AUD.

Figure 4. Average (±SE) ERP P3b amplitude for non-alcohol cues (target), alcohol cues (target), and household cues (standard) in Minimal and Pronounced AUD groups.

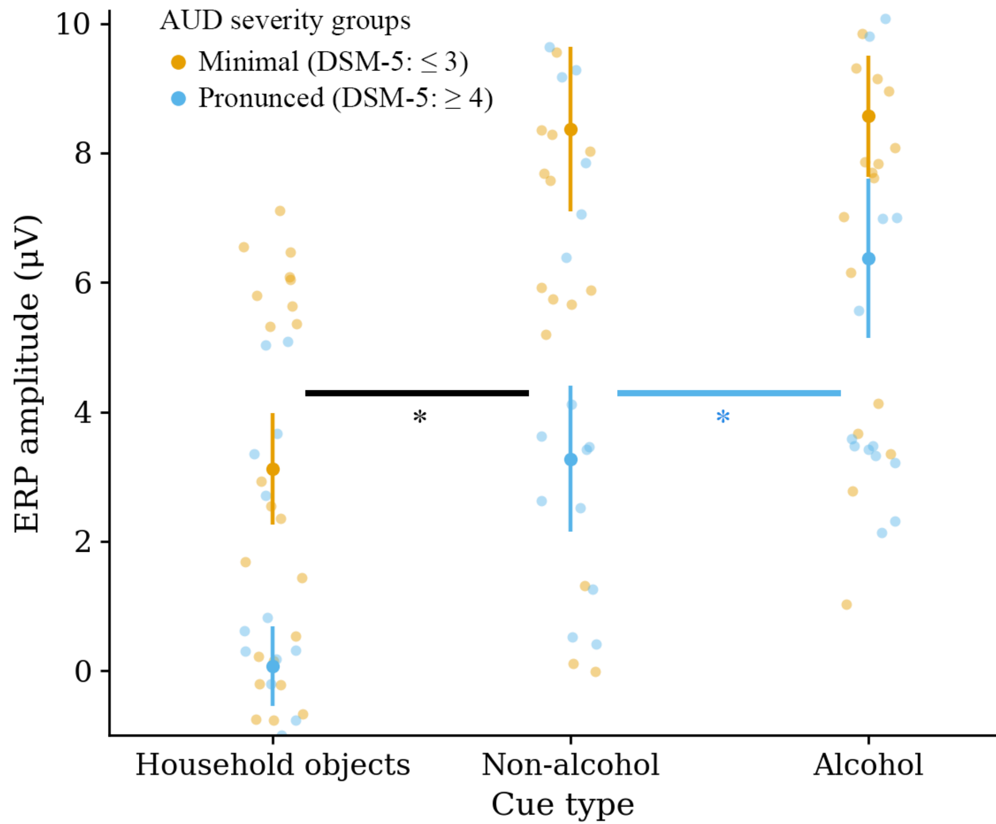
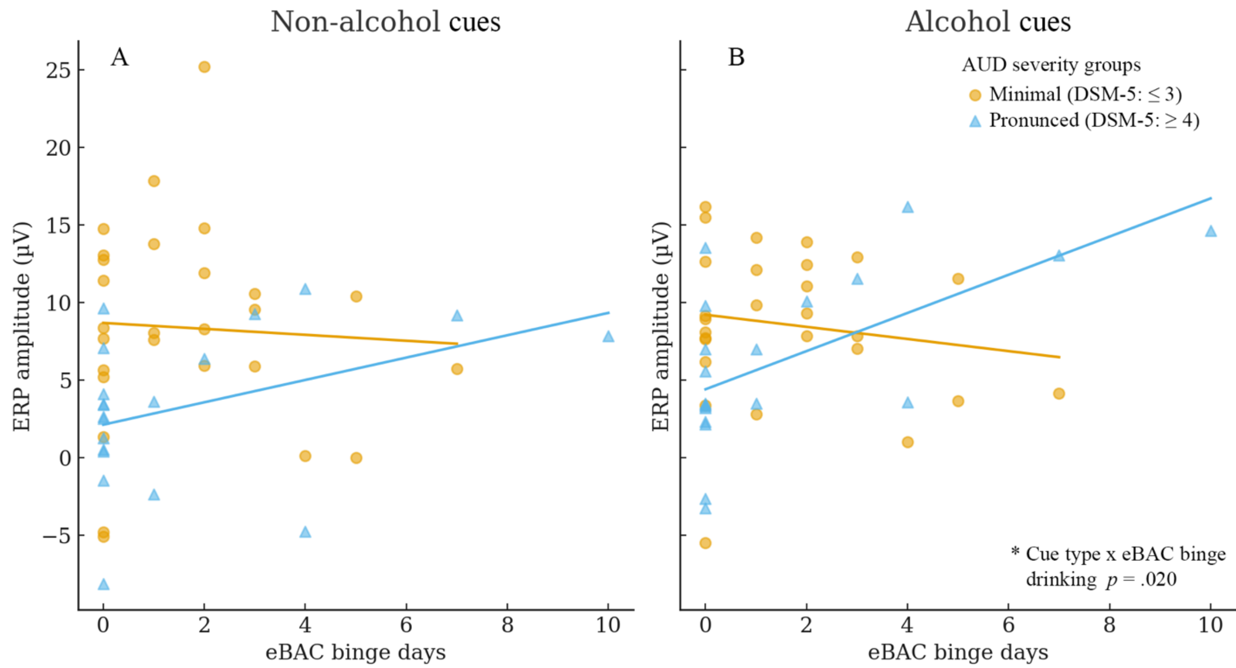
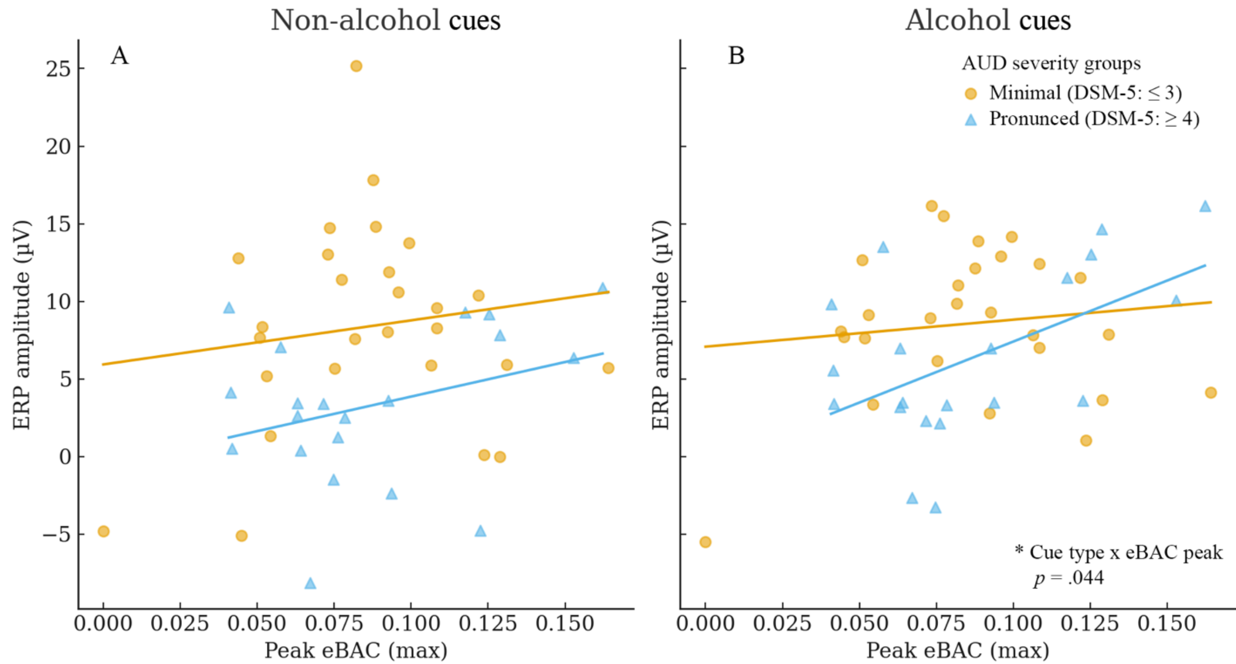


Figure 5. ERP P3b amplitude (μV) as a function of eBAC-defined binge days.



Note: Group-specific trends are shown for Mild vs Pronounced severity (DSM severity: 0–1 = Mild; 2–3 = Pronounced). The left panel (A) displays non-alcohol cues and the right panel (B) displays alcohol cues.

Figure 6. ERP P3b amplitude (μV) plotted against the highest peak eBAC for each participant during the 14 days.



Note: Group-specific trends are shown for Mild vs Pronounced severity (DSM_severity: 0–1 = Mild; 2–3 = Pronounced). The left panel (A) displays non-alcohol cues and the right panel (B) displays alcohol cues.